

(FILE 'HOME' ENTERED AT 10:57:26 ON 21 JUN 2001)

FILE 'BIOSIS' ENTERED AT 10:57:34 ON 21 JUN 2001

L1 42 S OOCYTE? AND (NO DONOR OR NITRIC OXIDE DONOR OR NOS OR NOS  
IND  
L2 159342 S FERTIL?  
L3 12 S L1 AND L2  
L4 14 S OOCYTE? AND (NOS OR NITRIC OXIDE SYNTASE) AND INHIBIT?  
L5 4 S BOVINE AND OOCYTE AND (NITRIC OXIDE)  
L6 6 S BOVINE AND OOCYTE? AND (NITRIC OXIDE)  
L7 26 S HUMAN AND OOCYTE? AND (NITRIC OXIDE)

FILE 'CAPLUS' ENTERED AT 11:35:31 ON 21 JUN 2001  
L8 19 S HUMAN AND OOCYTE? AND (NITRIC OXIDE)

FILE 'MEDLINE' ENTERED AT 11:40:51 ON 21 JUN 2001  
L9 26 S HUMAN AND OOCYTE? AND (NITRIC OXIDE)

L4 ANSWER 5 OF 14 BIOSIS COPYRIGHT 2001 BIOSIS  
AB This study was conducted to determine whether endothelial-derived nitric  
oxide synthase (eNOS) affects meiotic maturation of mouse **oocytes**  
in vitro. Cumulus-**oocyte** complexes (COC) were isolated from  
ovarian follicles of 27-day-old PMSG-primed wildtype (WT), and  
eNOS-knockout (eNOS-KO) females, and cultured in drops of medium under

oil  
at 37degreeC for 16-18 hr. Experiment 1 was carried out to determine  
effects of eNOS deficiency on the ability of COC to mature in vitro. To  
determine whether acute synthesis of nitric oxide (NO) was required for  
**oocyte** maturation, COC collected from WT mice were cultured in  
medium without (control) or with different doses of Nomega-nitro-L-  
arginine methyl ester (L-NAME), an **inhibitor** of NOS  
(exp. 2). To assess effects of NO deficiency on the kinetics of germinal  
vesicle breakdown (GVBD), COC from WT and eNOS-KO females were observed  
for 3.5 hr. COC from WT females were also incubated in medium without or  
with L-NAME (exp. 3 and 4). After the culture period, cumulus cells were  
removed, and **oocytes** were counted and classified as metaphase II  
(M II), metaphase I (M I) or showing atypical (degenerative) morphology.  
To determine viability and nuclear morphology of **oocytes**, they  
were stained with fluorescein diacetate or 4,6-diamidine-2'-phenylindole  
dihydrochloride, respectively. There were no differences in body weights  
but ovarian weights were lower in eNOS-KO mice compared with WT mice (P <  
0.05). Ovaries from eNOS-KO mice contained fewer COC collected relative

to  
WT mice (P < 0.01). Maturation of COC from eNOS-KO mice or WT  
**oocytes** treated with L-NAME resulted in a lower percentage of  
**oocytes** at M II stage (P < 0.01 and P < 0.05, respectively) and a  
higher percentage of **oocytes** at M I or atypical stages compared  
with those from WT (P < 0.01 and P < 0.05, respectively). Many  
**oocytes** that showed either an arrest in M I stage or abnormal  
morphology were not viable. Several **oocytes** in M II stage  
demonstrated abnormalities in distribution of maternal chromosomes. Our  
data demonstrate that eNOS-derived NO is a key modulator of **oocyte**  
meiotic maturation in vitro. These results support our previous  
observations in vivo and indicate that eNOS/NO has independent functions  
in both **oocyte** maturation and follicular/**oocyte**  
development.

ACCESSION NUMBER: 2000:167668 BIOSIS  
DOCUMENT NUMBER: PREV200000167668  
TITLE: Nitric oxide is essential for optimal meiotic maturation  
of murine cumulus-**oocyte** complexes in vitro.  
AUTHOR(S): Jablonka-Shariff, Albina; Olson, Lisa M. (1)  
CORPORATE SOURCE: (1) NCP-U4B, Monsanto Company, 800 N. Lindbergh Blvd.,  
Saint Louis, MO, 63167 USA  
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LANGUAGE: English  
SUMMARY LANGUAGE: English

*fab date*

L1 ANSWER 5 OF 42 BIOSIS COPYRIGHT 2001 BIOSIS  
 AB We investigated the extent to which NO participates in the developmental competence (**oocyte** maturation, fertilization and embryo development to blastocyst) using an in vitro culture system adding sodium nitroprusside (SNP), **NO donor**, and **NOS** inhibitor (N-omega-nitro-L-arginine methyl ester, L-NAME). We also assessed the effects of NO/**NOS** system on blastocyst implantation using an in vitro trophoblast outgrowth assay. The treatment of low concentrations of SNP ( $10^{-7}$  M) significantly stimulated meiotic maturation to metaphase II stages in cumulus enclosed **oocytes**. In contrast,  $10^{-3}$  and  $10^{-5}$  M L-NAME demonstrated a significant suppression in resumption of meiosis. This inhibition was reversed by the addition of SNP. No development beyond the four-cell stage was observed by the addition of high concentration of SNP ( $10^{-3}$  M). Inhibition of embryo development, especially the conversion of morulae to blastocysts, was also observed in the treatment of lower doses of SNP ( $10^{-5}$  and  $10^{-7}$  M). Similarly, inhibition of NO by **NOS** inhibitor resulted in the dose-dependent inhibition of embryo development and hatching rates, but the concomitant addition of SNP with L-NAME reversed the inhibitory effect by each SNP or L-NAME treatment. Furthermore, low concentration of SNP ( $10^{-7}$  M) but not high concentration of SNP ( $10^{-3}$  M) significantly stimulated trophoblast outgrowth, whereas the addition of L-NAME suppressed the spreading of blastocysts in a dose-dependent manner. These results suggest that NO may have crucial roles in **oocyte** maturation and embryogenesis including the process of implantation. The observed differences in required amount of NO and the sensitivity to cytotoxicity of NO in each developmental stage embryos may also suggest that NO/**NOS** system is tightly regulated in developmental stage specific manner.

ACCESSION NUMBER: 2001:129039 BIOSIS  
 DOCUMENT NUMBER: PREV200100129039  
 TITLE: Requirement of nitric oxide for murine **oocyte** maturation, embryo development, and trophoblast outgrowth in vitro.  
 AUTHOR(S): Sengoku, Kazuo (1); Takuma, Naoyuki; Horikawa, Michiharu; Tsuchiya, Keiko; Komori, Harumi; Sharifa, Dinara; Tamate, Kenichi; Ishikawa, Mutsuo  
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